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THE UNIVERSITY OF TORONTO MEDICAL JOURNAL

(An Undergraduate Publication)

This journal is published seven times a year, November to May, by the Medical Society of the University of Toronto, to serve as a medium for student expression and to bring to the student recent articles of scientific interest.

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Volume XI TORONTO, JANUARY, 1934 Number 3

EDITORIAL

LICENSING OF PRACTITIONERS.

Whenever the question of Medical Council examinations and the payment of fees for a license to practise comes up we recall the history of Medicine in Canada and the incidents which led up to the formation of the Medical Board. In the early days of Upper Canada, about 1783 when thousands of United Empire Loyalists began to settle in Canada, there was a great scarcity of qualified medical practitioners. Most of those practising in Canada were Military and Naval doctors, very few having come from England to establish practices. The need for doctors was aggravated by the fact that many of the U. E. Loyalists scattered into outlying parts to establish their new homes. As a result the country soon became over-run with self-made physicians, many of whom had come over from the United States with the sole object of making money, having little or no knowledge of medicine nor even of the principles of Anatomy, Biology or Physiology, and many being generally ignorant to an extreme. Their general custom was to employ two drugs —opium and mercury. If the patient was suffering with pain of any kind opium was given. If there was some doubt about the disease—calomel was the remedy. Wm. Canniff, in his book on the Medical Profession in Upper Canada, quotes a story told by Bishop

Strachan, who was greatly interested in securing some form of legislation for the control of medical practitioners in this country. Bishop Strachan says:

"I was lately visiting a young woman ill of a fever. The Doctor came in, felt her pulse with much gravity, pronounced her near a *crisis*. 'She must take this dose,' said the gentleman, pouring out as much calomel on a piece of paper as would have killed two ploughmen. 'Pray, what is this?' said I. Doctor: 'A febrifuger!' "Is it not calomel?' 'Yes.' 'You mean to divide this into several doses?' 'Not at all.' 'But the patient is weak.' 'No matter, I likes to scour well.' 'Do you not weigh carefully so powerful a medicine before you give it?' 'No, sir, I knows exactly!' "

Another story told of this time speaks of a bill sent to Mrs. John Gould by a self-taught physician.

"The Estate of Mrs. John Gould. Dr.

"To Dr., For medsin and attendants whene he was choked with a large peas of Butter, no of meat, £3."

Another refers to a bill sent in by a doctor, with a deduction of £6, for "killing your son." This was because the doctor had carried small-pox to the son, who died of it.

Little wonder, then, that, in the face of such tales as these, the need for a medical licensing board became acute. In England, too, some two centuries before, apparently a similar state of affairs existed. Of course at this time many ills were treated by housewives remedies handed down in folk-lore, some of which doubtless contained drugs which we use to this day. But there were, evidently, numerous quacks who preyed upon a credulous populace doing much harm by the misapplication of the few principles of treatment which they knew and among the supporters of organized qualified practitioners was John Hall, a surgeon of London, who wrote in 1565: "An Historiall Expostulation Against the beastlye Abusers, both of Chyrurgerie and Physyke, in oure time, with a goodlye Doctrine and Instruction, necessarye to be marked and followed, of all true Chiurgiens."

In this way, the beginnings of medical licensing boards were established and no one should attempt to deny the value of such boards even at the present time. As far as the Medical Council of Canada is concerned, we hope the day is not far distant when students of Toronto and other large medical schools will not be required to write a set of Council examinations in addition to their own University exams. As for the fees required to be paid to the Medical Council of Canada, these seem to us to be exhorbitantly high, not forgetting that we must be grateful for the fact that our University fees are lower than those of most of the medical schools of the same standing as Toronto. We feel that students who are allowed to graduate from the University of Toronto in medicine should, ipso facto, be allowed to practice with little or no further difficulty to overcome.

MEDICAL SOCIETY NOTES.

Open Meeting.

The first open meeting of the Medical Society was held on Wednesday, December 13th, Dr. Fitzgerald being the speaker. This meeting was in the nature of a send-off for the Dean, being held just before his departure for a trip to India as a delegate of the Rockefeller Foundation. Dr. Fitzgerald gave us a very interesting half hour with numerous reminiscences of his earlier work in the production of antitoxin and the development of the Connaught Laboratories, and without attempting to moralize too strenuously, concluded with a few practical words of advice.

Daffydil, and Medical At-Home.

Plans for these two functions are going ahead, and with very able committees in charge of both our anticipations for two evenings of pleasure should be fully realized.

OBITUARIES

DR. FREDERICK ADAM CLELAND.

DR. FREDERICK ADAM CLELAND, a leading figure in surgical practise in Toronto, died suddenly on November 26, in his sixtieth year, from coronary thrombosis. He had suffered two previous attacks, but was able to conduct his practise until the last.

He came of a family influential in business and public affairs, and was born and prepared for matriculation in Meaford, Ontario. Entering the University of Toronto in the Faculty of Arts, he lived in the residence in the North wing of the Main Building, since abandoned, but famous in its day for the powerful life-long friendships made within its walls. After four years, full of vigorous interest in all student activities, including a place on the cricket eleven and captaincy of the lacrosse team, he entered medicine with a mind well matured, and graduated in 1901 with the silver medal.

Desirous of post-graduate work,. and without funds, he then followed the suggestion of Meaford friends living in New York City, and hung out his shingle on West 42nd St. He built up a large general practice, which he maintained throughout his stay there. Practical in the affairs of life, he managed his time rigidly, in order to give assiduous attention to the hospital work which was his goal. Obtaining posts in the Surgical Dispensary of the New York Post Graduate Hospital, and as Demonstrator of Anatomy in the New York Polyclinic, he worked up gradually to be an assistant in Gynaecology under Taylor, at the Polyclinic, and decided to make this his special life work. He continuously spent much time in operative work on the cadaver, and always valued greatly the facilities for this work in the big centres, and in later life made many short trips to New York or Chicago for rapidly perfecting himself, by this means, in some new technique.

Having meanwhile married Miss Anna Goldie of Ayr, Ontario, thoughts turned homeward, and in 1909 he returned to Toronto to endeavour to establish himself as a gynaecologist and abdominal surgeon. This second experiment of undertaking a new venture with slender financial resources met with success also, and the rapidity of his rise to a responsible position in practice was remarkable. The qualities which contributed largely to this were, a quick sympathy with the viewpoint of the patient, excellent judgment in planning action; courage and quickness in changing plans during an operation; and detailed post-operative care, begotten of years spent in general practice His aperative technique was finished.

In public medical life, he first received a teaching appointment in Gynaecology and Obstetrics under Dr. Frederick Fenton in St. Michael's Hospital, and succeeded him in charge of this work. He was later asked to form a gynaecological service in Grace Hospital, and transferred his university appointment to the surgical service of Professor Cameron at the Toronto General Hospital. Here he worked until 1922, when he was appointed assistant professor of Gynaecology and Obstetrics, relinquishing this post owing to impaired health in 1928.

He was always active in the affairs of the Academy of Medicine and rose to the presidency. During his term in the latter office the outstanding events were the visits of Lord Dawson and Sir Ewan MacLean of Cardiff, with the dramatic announcement to the medical world, by Blair Bell, of his lead treatment of cancer.

He was elected fellow of the Association of American Gynaecologists, Obstetricians and Abdominal Surgeons in 1926, and was twice president for their meeting in Toronto in 1928.

In 1918 he went as Senior Surgeon with the Canadian Expeditionary Force to Siberia.

In private life the loyal maintenance of old friendships and the pleasures of travel occupied his time. A martyr to hay-fever, he always took a long Autumn holiday, often a trip abroad, and in mid-winter in later years he went South for three or four weeks. His steady and deep pleasure was his enjoyment of family life, and in this he was singularly blessed.

He is survived by his widow, two sons and two daughters.

H. S. Hutchison, M.B.

Just as we go to press we learn of the sudden death of Dr. Henry S. Hutchison, Assistant Professor of Medicine, on Saturday, January 6th, at the age of 55. Dr. Hutchison had been seeing patients in his office on Saturday afternoon, and not feeling well, had gone home early, passing away in a very short time. Dr. Hutchison had contributed numerous articles to this Journal, of which the latest was the biographical sketch in this issue, of Dr. Cleland, with whom he had been closely associated. His death will be mourned by a wide circle of relatives, friends and associates.

JOHN CHARLES HENRY COPP.

SINCE our last issue, the University has suffered a great loss in the passing of Johnny Copp. Throughout his school and university career he has been an outstanding character.

He received his preliminary education at Model School, where he was a leader in games and he continued his education at the University of Toronto Schools. His career there was outstanding and he received practically all the honors that could be bestowed on a student. He represented his school in both rugby and hockey and was captain of the former team. He also won the Nesbitt Gold Medal for general proficiency, ability and athletic prowess, as well as the Rowell Scholarship for general scholastic attainments. He was president of the Athletic Association and was elected school captain, this being the highest honour that the Schools can award.

Since entering the Faculty of Medicine at the University, his career has continued to be marked with successes. During his first year he was elected to the position of vice-president of his year and represented his university on the O.R.F.U. football team and the Senior B hockey team.. For the last three years he has been an outstanding member of the Intercollegiate football team. This year he was honoured by his fellow-undergraduates by being elected to the House Committee at Hart House.

Outside interests also claimed his attention as he was an accomplished musician and leader of his own orchestra. One of his greatest joys was his association with younger boys as Head Councillor of Camp Cayuga and leader of a Sunday School Class at Rosedale United Church. His fraternity was the Alpha Delta Phi.

To quote the words of our President, the Rev. Dr. Cody, "Few undergraduates have won such a place in the affection and esteem of their fellows as has John Copp. He bore himself 'without fear and without reproach.' His life and character are a challenge to us who remain."

H. A. Henderson.

CHRONIC ULCERATIVE COLITIS

E. J. MALTBY, M.B., M.A., F.R.C.P. (C.)
Toronto General Hospital.

Definition.

Chronic ulcerative colitis is a disease characterized by ulceration of the large bowel, the primary cause of which is not known, but has as secondary invaders different bacteria; there is a marked tendency for relapses and remissions.

History.

Ulcerative colitis was described first by Wilks and Moxon in their book on pathology in 1875. Ten years later Allchin exhibited before the London Pathological Society the first typical colon, showing changes due to this disease. In 1907 the use of the proctoscope and sigmoidoscope in the diagnosis of ulcerative colitis and other diseases of the rectum and colon was advocated by Lockhart-Mummery. From about 1907 to 1920 most interest was centred about the clinical course of the disease.

Aetiology.

A large group of individuals consider the condition to be due to infection in which the usual bacterial inhabitants of the large bowel play an aetiologic roll; a few others feel that the bacillus of dysentery is the causative agent. Rolleston considers that the disease is primarily some defect in the mucosa of the colon, with the normal intestinal inhabitant taking on pathogenic characteristics. Bargen thinks that the condition is caused by a specific organism — a diplococcus.

Pathology.

Ulcerative colitis is a disease of the colon presenting general manifestations in the patient. One's understanding of the pathological changes in the colon can best be obtained by repeated rectal and sigmoid examination, for if one depends on autopsy material, one sees the end stage of the disease only. At the onset the rectal mucosa is usually the first part to be obviously affected; one sees a somewhat reddened, slightly oedematous mucosa which bleeds on the slightest trauma. Within a short time, here and there small submucosal abscesses are found, but more often superficial ulcers covered with a greyish exudate. Depending upon the severity, the size of the ulcer varies greatly; in some cases it is difficult to find the normal mucosa, which appears only as small islands on a greyish surface. Occasionally the submucosal surface has a red appearance, making it quite difficult to recognize the mucosa left. Another type of response to this condition is a very marked oedematous reaction of the mucosa and submucosa so that the lumen is greatly encroached upon, with the wall a fiery red. At the onset of a remission the mucous membrane becomes less red, the ulcers heal over, and there is less tendency to bleed. At this stage, or at the onset of the attack, the mucosa often shows a granular appearance. After a few months the bowel may look normal. If the disease has been severe or prolonged, there may be small polypoid projections of the mucosa consisting of mucous membrane, the base of which has become pinched by contracting fibrous tissue causing it to project into the lumen.

Sometimes the inflammatory reaction in the bowel wall is sufficient to produce permanent stricture of the colon; in this case the wall is thickened considerably and stricture extends for a half inch or more along the gut. The ulceration may involve a moderate sized vessel causing considerable haemorrhage. Only rarely does the ulceration extend through the muscular and peritoneal coats, causing perforation and a peritonitis. Another rare com-

plication is ulceration from the rectum into the vagina.

Ulceration of the mouth, tongue and pharynx may occur during a very acute exacerbation of symptoms, but dehydration alone is not the cause, for one never sees this with the dehydration of diabetic coma. Multiple subcutaneous abscesses are occasionally met with, and sometimes moderately extensive superficial sloughing of the skin. In their book, "The Colon, Rectum and Anus,' Rankin. Bargen and Buie show an excellent illustration of such a condition.

In our experience, arthritis, at some time in the patient's illness, has been quite common. It usually does not accompany every exacerbation, and sometimes assumes the true rheumatoid form. Erythema nodosum is a rare complication.

Clinical History and Physical Findings.

Variations in the history and findings are quite considerable. For the purpose of simplification these cases may be classified roughly into three groups:

A. Acute fulminating ulcerative colitis.
B. Non-remitting ulcerative colitis, subacute and chronic.
C. Remitting ulcerative colitis, subacute and chronic.

A. **Acute fulminating ulcerative colitis.** This type of case presents a history of relatively short duration. Usually the patient has been ill a few weeks before seeing a doctor. When he is seen he is bedridden, and by this time there is considerable pallor of the skin. The bowel movements may be as frequent as twenty to twenty-four a day, and the stools consist chiefly of blood, pus and mucus mixed intimately with the faeces. Abdominal cramps may be quite severe, and it is common for incontinence of faeces to be present. The patient quickly shows emaciation, anaemia and dehydration, although, curiously enough, the tongue seldom shows much evidence of this. Water by mouth may cause increase in the diarrhoea. Ingestion of food often provokes an immediate desire for stool. The temperature is elevated and may be swinging in type, but occasionally one sees a very acute ulcerative colitis case afebrile. The pulse rate is rapid, often out of proportion to the temperature. A continued rapid pulse is of serious import.

On examination of the abdomen moderate distension is found, and on palpation there is a doughy sensation with generalized tenderness. Frequently one can feel some portion of the colon as a firm cord-like structure. Ulcerative colitis never seems to cause noticeable enlargement of the spleen.

Sigmoidoscopic examination shows an acutely inflamed mucosa with different sized superficial ulcers. Occasionally the bowel may be almost completely denuded of mucous membrane, leaving a greyish surface covered with exudate. Haemorrhage may be quite profuse in this type of case.

X-ray of the colon shows a loss of haustral markings, and in some cases a feathery or irregular surface is shown, denoting ulceration of the mucosa.

These patients die within a year, but usually live about two months at least.

Differentiation of this type of ulcerative colitis from dysenteric colitis may be quite difficult, but the points of differentiation are: by isolation of the Shiga or Flexner organisms; presence of a high agglutination titer for the above organisms; and the epidemic character of dysenteric colitis. Amoebic dysentery is not such a problem in the temperate zone. The crucial test is the demonstration of amoebae in the stool.

B. **Non-remitting ulcerative colitis, subacute and chronic.** In this type the patients may continue with three to five bowel movements for three, four and five years, and yet be able to carry on with their work, suffering only from increased fatiguability. They often give the history that whenever they

contract some mild infection, such as an upper respiratory infection or have a dental extraction, a disturbance in the bowels occurs with blood and pus in the stools. These patients have a pale, sallow appearance, even though the haemoglobin is about normal. The abdomen may show some distension, with gurgling sensation on palpation. The stool will show varying amounts of blood, pus and mucus.

Sigmoidoscopic examination reveals either the granular appearance of the mucous membrane or definite ulceration; in either case the mucous membrane bleeds readily. X-ray show frayed edges to the colon. The colon will have the typical sausage shape lacking in haustra and having varying width, depending on the amount of fibrosis in the bowel.

C. **Remitting ulcerative colitis, subacute and chronic.** Again one finds a great variation in the course of the disease. Some patients have only a few relapses with short remissions before they succumb, while others have a number of relapses, these being relatively short and mild, with long intermissions. One case reported in the literature died from ulcerative colitis thirty-six years after the primary attack. I think the cases we see have, at the outside, about ten to twelve years history from the onset of the disease to the termination. The remitting ulcerative colitis type is the commonest type we see here.

During remissions the bowel movements may return to normal number and become normal in consistency, with no evidence of blood, pus or mucus. True enough, these people say that if they work to the point of fatigue, suffer from some mild infection, or indulge in too rough a diet, they will have a short period of frequent fluid stools, the length of upset depending on the duration of the inciting cause. Other patients during the remission will have two or three rather loose stools each day, but become so used to this that they consider it quite normal.

Examination of the patient during the remission will reveal little of note except that on sigmoidoscopic examination one may find healed ulcers as small superficial scars or, here and there, pseudopolypi projecting from the mucous membrane. During the relapse they simulate the other types in every way.

Complications.

One of the most common complications is intestinal haemorrhage. It occurs during the acute stages of the relapse and is usually preceded by bloody stools. It is apt to be repeated and may be quite grave.

Arthritis is another common complication. As a rule, too, this occurs during acute phases of the disease. The type is usually the subacute infective arthritis, but we have had one typical rheumatoid arthritis occurring three years after the onset of the colitis. There are no particular joints affected, although arthritis is much more common in the extremities than in the spine.

Superficial ulceration of the skin in the perineal region may occur during the acute stages The ulcers show slight inammatory reaction and heal leaving little scarring. Similar, but usually more widespread, ulceration may occur on the back or legs.

Stricture of the bowel is not very common. Considerable constriction of the bowel can occur without obstruction due to the fluid stools. X-rays demonstrate this condition best, but one must not be misled by temporary segmented spasm of the colon. Perforation occurs occasionally, and due to the low resistance of the tissues, is almost invariably fatal.

Ulceration of the tongue and mouth occurs, but other than giving an indication of the severity of the toxaemia, is of little significance in itself. Migrating thrombophlebitis also occurs.

Treatment.

The cause of ulcerative colitis is unknown, so that treatment is wholly symptomatic.

Rest. All patients with ulcerative colitis should be told that their period of invalidism is of long duration, that the stay in bed will probably be for months and, preferably, it should be in hospital. When the fever and activity in the bowel have receded, he is allowed up and about, but not back to work for at least a month. The patient must regulate his work so that he can avoid fatigue. He must spend eight hours of the twenty-four, or more, if possible, in bed.

Diet. The diet should be simple and low in residue, vegetables in the form of purees, the carbohydrates moderate in amount and the total caloric intake six hundred to a thousand calories above basal diet. Leafy vegetables, pickles, spices, nuts, fresh salads, etc., are to be avoided. Milk as the chief source of diet is frequently poorly tolerated.

Laxatives.—Sodium sulphate is used during the active stage of the disease in doses of one ounce once or twice a day. During remissions sodium sulphate or paraffin oil is used to keep the stools soft.

Local treatment to colon. Colonic irrigations are used, but most patients tolerate them poorly. If any are used, the milder the better, such as normal saline or isotonic sodium bicarbonate. Various astringent solutions have been used, such as: silver nitrate, potassium permanganate, and tannic acid solution.

Sedatives. Various sedatives are used to decrease bowel activity during the acute stage of the disease. Morphine is used frequently either as lead and opium pills or as starch and opium enemata. Seldom is this form of therapy used here. Sometimes it is necessary to give sedatives to control the pain from the cramps and codeia by hypodermic injection will usually control it.

Intravenous therapy. Dehydration from diarrhoea is best controlled by intravenous administration of fluid. Blood transfusions have a remarkable effect at times on these patients. Frequently considerable ketosis is found in the markedly toxic and dehydrated patients: both the dehydration and ketosis are controlled by intravenous administration of 5 per cent. glucose in saline.

Vaccine and serum therapy. Hern, in comparing different modes of treatment, found that the mortality over a certain number of years was less with the use of anti-dysenteric serum than with other forms of treatment, but he thought that due to an insufficient number of cases this conclusion might be erroneous. Hurst uses this method of treatment a great deal. The standard amount is about 300 cc. of polyvalent serum: 40, 60, 80 and 100 cc. given intravenously either on consecutive or alternate days. Intramuscular injection may be used, but only 10 cc. is administered for several days. The results from the use of Bargen's vaccine and vaccines prepared from the patients' stools are difficult to assess because of the marked tendency for remissions in these patients but, on the whole, are disappointing.

Surgical intervention. Appendectomy:—This procedure is of relatively little value although advocated by some. If irrigation of the colon by the appendix were efficacious, then colonic irrigation should be of equal value since it has been shown that in this condition a barium enema quickly reaches the caecum. Therefore, why subject the patient to the danger of an unnecessary operation?

Caecostomy:—This operation is similar in effect to appendectomy and since it diverts little of the faecal stream it has the same drawbacks.

Sigmoidostomy:—This procedure may allow for healing of the distal portion of the colon, but it must be remembered that frequently the whole colon is affected and the proximal end may show considerable inflammatory reaction It has been shown, moreover, that the colon may appear normal to

the naked eye, yet on section show a diffuse round cell submucosal inflammation. One still must face the problem of the proper time of closure, and since a relapse may be just around the corner, this is difficult.

Ileostomy:—One feels this to be the most logical operation. Again you are faced with the permanence of the operation. Cases are on record where the patients have gone five or six years with ileostomy without suffering any symptoms, but when continuity of the gut was re-established the whole ulcerative colitis process became activated. Evidently there is no doubt that once the patient has recovered from the immediate effect of the operation there is an improvement in the general health.

Colectomy:—This operation is fraught with a high mortality and one accomplishes in most cases as much with ileostomy except where the bowel must be removed as in cases with malignant degeneration of mucous membrane of the colon.

The complications affecting the colon, such as: stricture, perforation, and fistulae, are treated on their own merits.

In conclusion, one is impressed with the gravity of ulcerative colitis and with the paucity of knowledge of the underlying factors predisposing and causing it.

OBITUARIES

DR. IRVING HEWARD CAMERON.

ON Friday, December fifteenth, the death occurred in his 79th year, of Irving Heward Cameron, M.B., LL.D., Edin., F.R.C.S., Eng., Edin., Ireland, formerly Professor of Surgery in this University from 1897 to 1920, latterly Professor Emeritus. Few of the present number of under-graduates have had the pleasure of knowing Mr. Cameron—as he preferred to be called—but many of our clinicians have related stories of their former Professor which leave no doubt in our minds as to how greatly he was admired and respected. None of his students will readily forget his lectures which must have been among the most enjoyable they attended and from which many extracts have appeared from time to time in this journal.

The passing of Mr. Cameron deprives the Medical profession in Canada of one of its most distinguished members.

DR. THOMAS VERNER O'BRIEN WILSON.

HIS many friends and acquaintances in medicine were shocked to learn of the death of Thomas Verner O'Brien Wilson, M.D., on Nov. 30th last, in his 27th year. Tom had been an interne at the Sick Children's Hospital for the last few months, having previously spent a year at the Royal Alexandra Hospital, Edmonton, following his graduation from Toronto in 1932. He was the son of Mr. and Mrs. E. V. Wilson of Shanty Bay, and grandson of the late Colonel W. E. O'Brien. Some days before his death he contracted a severe septic throat condition from which pneumonia developed.

VOLKMANN'S ISCHAEMIC CONTRACTURE

KEITH P. BONNER, '34.

In 1667 Stenson, a Florentine physiologist, first called attention to a paralysis affecting the posterior extremities of animals following ligation of the abdominal aorta. This fact rested upon its laurels for two hundred years, and was reaffirmed in 1869 by Schiffer who, admitting the paralysis, did not believe that the ischaemia in the extremities was the basic etiological factor in the condition.

In 1875 Volkmann described a severe paralytic contraction of the hand which he ascribed to tight bandaging in the treatment of fractures in the neighbourhood of the elbow-joint. In 1881 he published another article upon this subject, in which he reaffirmed his belief in ischaemia as the cause of the contracture. He further qualifies his statement, inasmuch as he divides the ischaemia into two factors, viz.: deficient arterial supply, and obstructed venous drainage. He also calls attention to the fact that contraction and paralysis occur simultaneously, whereas in lesions of the nerve, paralysis is early in appearance, while contractions appear later.

This historical survey is of assistance in isolating the condition clinically, as it is customary to-day to describe practically all cases of contracture following the application of a tight bandage, tourniquet, or splint, as Volkmann's ischaemic contracture.

Pathological Considerations.

Ischaemic contracture is a deformity found principally in children between one and fifteen years of age, though it may occur at any age. It most commonly follows injury to the elbow, and is frequently, but not always, associated with fracture. The upper extremity is involved in more than ninety per cent. of cases. In common with fractures, it is more often seen in males.

In consideration of the exciting cause, the literature offers many theories based upon conjecture and remarkably few upon experimental evidence. It was not until Barney Brooks of St. Louis in 1922 published his report of experimental investigations of pathological changes in muscle as a result of circulatory disturbances that the former theories could be revised from a scientific standpoint. Brooks' investigations led him to differentiate between the pathological conditions produced in muscle as the result of arterial lesions and venous lesions of varying degrees. His findings may be summarized as follows:

1. Permanent arterial occlusion may show:
   a. no demonstrable anatomical or physiological change.
   b. a temporary loss or depression of muscular function followed by a progressive improvement leading to a complete restoration of function with no demonstrable anatomical changes.
   c. the development of a sharpely demarcated gangrene.

2. Temporary arterial occlusion may show:
   a. no demonstrable alteration in the affected muscles, or
   b. if the obstruction is of long duration, necrosis may result.
   A slight inflammatory change is sometimes seen in these cases, but there is no rapidly developing fibrosis and contracture.

These results apply equally well in muscles that have been isolated, thereby depriving them of any collateral circulation.

3. Occlusion of both artery and vein may show:
   a. no pathological change.
   b. necrosis of muscle, with complete absorption as the end result.

4. Nerve section with intact vascular apparatus may show:

paralysis of muscle with some degeneration, but no evidence of inflammation or fibrosis.

5. Injection of blood into the muscle substance showed, after a lapse of one month, no physiological changes or evidence of inflammation or fibrosis.

6. Venous occlusion leaving all other structures intact persistently showed the following changes on examinations made at intervals of from one to one hundred and one days, viz.:

Within an hour of operation the muscle became swollen, hard and dark blue in colour. Stimulation of the muscle directly or indirectly gave very little response. At the end of twenty-four hours there was marked extravasation of blood into the substance of the muscle and an accumulation of bloody fluid surrounding the muscle fibres. After forty-eight hours there was a marked collection of leucocytes about the muscle fibres. After ten days there was marked degeneration of the muscle fibres and an extensive proliferation of connective tissue extending into and involving all the surrounding structures, imbedding them in a dense fibrous mass.

It is unfortunate, perhaps, for purposes of comparison, that no other experimental work has been published upon this subject, so that any conclusions based upon facts already presented may, in the future, have to undergo considerable revision to conform with additional experimental evidence.

We may therefore conclude that the clinical picture of Volkmann's ischaemic contracture is essentially the resule of acute venous obstruction, and that ischaemia per se plays no part in the production of this condition. In ischaemia there is an acute anaemia produced which is incompatible with the process of an acute inflammation; and Brooks in his article further states that "any condition which develops within twenty-four or forty-eight hours and which is characterized by heat, swelling, pain and subsequent fibrosis, cannot be the result of permanent occlusion of an artery."

Practical application has been made of the fact that venous obstruction promotes a rapid proliferation of connective tissue in the clinical use of Bier's passive hyperaemia in cases where healing is deficient. It is unfortunate that no cases of Volkmann's contracture are reported from over-indulgence in this form of therapy.

Clinical Considerations.

Clinically, Volkmann's contracture is a rarity, and undoubtedly many contractures are given this classification in error.

Symptoms begin within a few hours after injury. The fingers become numb and swollen, and voluntary power is diminished or absent. The majority of patients will complain bitterly of pain in the whole arm, and on palpation the muscles are found to be firm in consistency. It is very easy to confuse these complaints with the discomfort resulting from injury and manipulation; and all patients exhibiting any of these symptoms should be carefully examined. Meyering lays stress upon the necessity of observing patients who complain of pain, especially if the pain persists in spite of morphine.

Some days after the initial symptoms have arisen, the swelling disappears and the muscles become hard and resistant; the muscle bellies normally soft and elastic are replaced by fibrous tissue, and contraction of the fibrous areas takes place.

The deformity of Volkmann's contracture depends upon the extent of muscle tissue involved. The flexor muscles of the arm are first and most severely affected. The fingers are flexed on the palm when the wrist is extended, and with the wrist flexed some degree of finger movement is often possible. Severe degrees of the condition may involve the extensor muscles of the arm as well as the small muscles

of the hand, so that the extremity is incapable of any movement below the elbow joint. Partial atrophy of opposing muscles not affected in the disease is likely from disuse, so that the end result is an immovable, grossly deformed and atrophic extremity.

In regard to treatment, as this condition is almost always associated with fracture, it should be remembered that what may be termed a bad reduction may give a good functional result, and poor reduction with good function is much to be preferred to perfect reduction combined with the disability of a Volkmann's contracture.

As soon as symptoms arise suggesting the possible onset of this condition, all bandages, splints or other apparatus should be removed, and the arm, supported on a pillow, should be exposed to dry heat supplied by an electric cradle. Should this not give relief, one must consider the advisability of surgically attempting to restore venous circulation. Such a procedure should not be undertaken lightly, as the incision converts a simple fracture into a compound one; also, swelling may be so extensive that suture of the wound may be impossible; and any infection superimposed upon the degenerate muscles is likely to result in gross and irreparable damage.

In an attempt to relieve the pressure in the muscles a small incision may be made in the skin followed by longitudinal splitting of the underlying fascia; a few observers claim to have used this procedure successfully in a small series of cases.

As a general rule our attempts to abort this condition once it has properly begun are ineffectual, and our surgical intervention may add considerably to the resulting disability. Hence the best treatment is prevention. This we can easily carry out; reduction of fractures should be delayed until the soft tissues are in condition to withstand the added insult of reduction and splinting. This combined with careful observation of all cases for the forty-eight hour period following injury and reduction may well forestall many disabilities and legal complications.

In the treatment of the established contracture many suggestions have been made, but it is useless to expect to get good function from an arm which has suffered such a loss of muscular tissue with interference of function evident in all other tissues.

Stretching on the shortened tendons is advised; this must be very gradual, as any traumatization of the muscle only causes an increased amount of fibrosis. Transplantation of tendons has found favour with some surgeons, while others prefer to transplant the sites of muscle origin. Still others shorten the radius and ulna in order to produce a relative lengthening of the shortened tendons.

Physiotherapy in the form of heat, massage and electrical stimulation is often useful in maintaining the maximum efficiency of the remaining functional tissues.

In reviewing all forms of treatment it is evident that prevention of conditions favourable to the establishment of contracture is the best form of therapy at present which can be recommended.

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CORONARY THROMBOSIS

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PART I.

Coronary thrombosis is a disease which, until the beginning of the century, was rarely recognized as a clinical entity, and little understood, but now is in the category of diseases which have a definite clinical picture and pathological background. The importance of this disease as one which causes death, sudden in many instances, also one which incapacitates individuals, is being more and more realized by clinicians and pathologists. The severe cases in which death occurs suddenly are of importance and interest to the coroner. The clinician, on the other hand, is not interested in these cases, because they are now readily diagnosed, the outlook is so grave, and treatment is of no avial. His interest lies in the milder cases of coronary thrombosis, which are more difficult of diagnosis, and which if recognized and with proper treatment have a more hopeful outlook.

It is thought by some that this disease is on the increase, but perhaps this is explained by the fact that more is known about it, and therefore it can now be properly diagnosed even in its milder forms. Also, the pathologists are more carefully examining the coronary arteries and their branches for the presence of thrombi.

Sudden deaths that were formerly attributed to "acute indigestion," because of the presence of severe epigastric pain with vomiting, are now known to be due to coronary thrombosis. Other cases were wrongly diagnosed as cerebral haemorrhage. It must be remembered, that death from this condition rarely occurs under twenty-four hours.

Of the ten chief causes of death for Ontario, organic heart disease heads the list, and has done so for the past ten years or more. It is seen that cardiac mortality in Ontario, 1921-1930, has increased from 115 to 170 per hundred thousand. Cardiac deaths are due to four main causal diseases, arteriosclerosis, rheumatic fever, hyperthyroidism and syphilis in order of total number. As cardiac deaths are registered in bulk, and not in terms of the underlying disease, consequently there are no reliable statistics available which show what percentage of deaths is due to any one of these four diseases. In the city of Toronto it is estimated that there are about 500 deaths a year, due to coronary sclerosis. Figures for coronary thrombosis are not available.

The historical development of our knowledge concerning the clinical recognition and association of clinical and pathological findings in coronary thrombosis forms an interesting chapter of modern internal medicine. Nowadays, the condition can readily be diagnosed, but it is only in the last few years that its distinctive features have been understood, recognized, and differentiated from those that pertained to allied but decidedly different pathological states.

Physiology of Coronary Circulation.

The coronary vessels receive a rich innervation from both the vagus and the sympathetic. The vagus conveys the constrictor fibres to the coronary arteries, and the sympathetic, the dilator fibres.

The coronary flow is increased by various conditions, anoxaemia, excess CO_2 in inspired air, liberation of adrenaline, increased cardiac inflow and increased blood pressure. A two-fold in-

crease in blood pressure results in a four-fold increase in coronary flow. During exercise the minute volume may increase ten times, i.e. as the cardiac output increases so does the coronary flow.

The coronary arteries are filled during diastole and early ventricular systole, and during ventricular systole the coronary artery flow is cut down to a minimum, attributed to compression of intramuscular branches of coronary vessels. The outflow from the coronary sinus is, however, greatest during ventricular systole, and is also increased to a less extent during auricular systole. It is estimated that 60% of the coronary flow returns to the heart chambers by way of the coronary sinus, the other 40% via the Thebesian vessels.

Pathology.

In coronary disease of the heart, the outstanding coronary lesion is coronary sclerosis. Coronary thrombosis always occurs on a previously sclerosed coronary vessel, according to Klotz; cases are reported in which thrombosis developed in absence of sclerosis, but these are extremely rare. No case of primary coronary thrombosis was encountered here, at Toronto General Hospital. The arteriosclerotic process brings about a stenosis of the vessel, but the complete occlusion of the vessel results from thrombosis. The sclerosis of the coronary vessel appears to have its beginning either in a primary endarteritis, or in an intimal degeneration followed by endarteritis.

Coronary Sclerosis. — The earliest signs of sclerosing lesions may appear in the 1st and 2nd decades in life, and then present themselves in increasing frequency with advancing age. The earliest lesions consisting of superficial fatty plaques have no effect on the coronary circulation, but represent the beginning of changes, which, with recurrent additions, give rise to serious distortions of the vascular function, as is recognized in chronic disease. This lesion, superficial fatty streak, is a progressive one, beginning early in life, reappearing at all ages, in association with acute infections and intoxications. The fatty substances are laid down in the intima by the phagocytic action of mononuclear cells, little or no fat being outside the cells, but with the degeneration of the phagocytes the fat becomes free, and seeps into the collagen of the connective tissue. At the same time a plaque is formed over the surface of the original fatty deposit by the overgrowth of the subendothelial connective tissue. This is the beginning of an atheromatous structure, which is always associated with chronic endarteritis.

There is another lesion similar to the above in the end, but having a different beginning. In young children, small nodules form in the intima, due to the presence of oedema and inflammatory cells. With exacerbations of the infection, reaction and proliferation occurs in the nodule, so that it gradually becomes larger and forms a permanent nodule. Later, a secondary process of degeneration involves the deeper layer of the intima, close to the media, in fatty and other degenerations.

So that atheroma, with its accompanying plaque, like endarteritis, may have its origin either in a primary fatty degeneration, or in a primary endarteritis as found accompanying acute infectious diseases.

During the 1st and 2nd decades of life the lesions of the intima do not encroach upon the lumen, whereas those appearing in the 4th decade occur more frequently and more rapidly, forming definite nodules which interfere with the circulation in the vessels.

Nodular endarteritis is the common type of lesion encountered in these vessels of the coronary system, which, as the process takes place in the intima, gives rise to a distortion and narrowing of the lumen of the vessel. The thickened intimal plaque, projecting into the lumen, outwardly pressing on the media, has an influence in bringing

about atrophic changes in the media, so that there is a narrowing of the media beneath the nodule. As there is no reaction to this process, there is not any compensatory growth of fibres in the media or adventitia. The enlargement of the nodule is possibly due to recurrent attacks of the original process that brought it about. This enlargement is formed gradually, and is made up of an overgrowth of intimal tissue nearest the lumen, and in the deeper part, a deposition of fat which forms the atheroma. The fat in this structure lies free, but also some of the connective tissue takes up the lipoids. The fat localizes in the deeper part of the intima by passing through the lymph channels from the lumen, or else by mechanical absorption of lipoids from the blood. In this chronic nodular endarteritis, the laminations of the intimal tissue remain intact, and so atheromatous ulceration rarely occurs. As the accumulation of lipoids goes on in the deeper layers, there comes a time when calcium salts begin to be deposited in the same area, and the final result is the formation of a hard, calcareous deposit, or calcified mass. There is also an extension of the chronic endarteritis laterally around the lumen of the vessel, so that the end result is more of an annular projecting mass, and the lumen is now very minute. This lesion always remains confined to the intima, the inner elastic membrane is involved in the atheroma; but except for atrophy of the media, there is no other change in the media or adventitia.

The narrowed lumen acts as an obstruction to the onward flow of blood, and in the dilated portion beyond the obstruction small eddies occur. These nodules produce more obstruction, if they occur in smaller branches, than if they occur in the main artery, for it needs only a little mass in the smaller artery. It is at these areas of endarteritis that partial or complete thrombosis is apt to occur.

These scleroses are dotted along the course of the right and left coronary arteries, at irregular intervals. The sclerotic patches are usually multiple; various branches of the coronary arteries being involved at the same time, although occasionally there are very few sclerotic areas. These patches are more common and occur more frequently in the left coronary artery, and the earliest lesions are seen in the anterior descending branch of the left coronary artery.

Changes Occurring in the Coronary Arteries.—The slow obstruction of coronary arteries is due to the progressive sclerotic process, chronic endarteritis, atheroma and calcification. While this process is going on, the anastomosis between the precapillaries and capillaries of the various branches of the coronary arteries is becoming more extensive, as there is a gradual dilatation of these vessels.

This process of sclerosis may begin early in life, but usually makes its appearance in the fourth decade of life. So that an older individual who has sclerosis of his coronary vessels has as well a better anastomotic collateral circulation than a younger individual. Thus he is able to withstand the shock of sudden occlusion due to thrombosis better than the younger person.

The sudden obstruction of the coronary arteries is due to thrombosis, less often to other conditions such as embolism. Thrombosis always occurs in a vessel with preceeding sclerotic changes. Thrombosis of these vessels is due, not entirely to changes in the vessel walls, but also to changes in the chemical constituents of the blood.

The histological appearance of fresh and of healed coronary thrombi corresponds with that of similar thrombi elsewhere in the body. Within twenty-four hours after the occlusion by thrombosis, fibroblasts and endothelial buds begin to invade the clot. In a few days, even before the red cells have undergone complete disintegration, fibrin has largely been replaced by connective tissue. The next stage is recan-

alization, which develops, either single or multiple, through the heart of the clot or else along its side, and the circulation is once more able to go on.

Recanalizaztions form not only in the occluding thrombi, but in granulations within thickened sclerotic vessel walls whose lumen is almost obliterated. During the time that the thrombus is being organized, collateral channels are carrying on the circulation.

In the examination of old healed infarcts and aneurysms, the occluded artery is seen to have undergone repair.

Factors in the Determination of Infarct Formation.—

1. *Degree.*—Sclerosis and occlusion of coronary artery reflects itself on myocardium. There are all variations to the amount of damage done to the myocardium, because of the impairment of circulation. There may be temporary anaemia, with no effect on myocardium at all, because collateral circulation is sufficient to re-establish the necessary nutrition. The most severe condition is infarct formation, due to the occlusion of a larger branch of one of the coronary arteries.

2. *Size.*—If a small branch is occluded, infarct formation may not necessarily result, but if the vessel be a large branch of one of the coronaries, then infarct formation will result.

3. *Time.*—The time factor is very important, because, if the occlusion takes place suddenly as in coronary thrombosis, a condition of acute myocardial degeneration results, with a consequent incompetency on the part of the whole heart. The end result may be softening, aneurysm and rupture. Whereas, if a coronary vessel is slowly narrowed and circulation is good, sufficient dilatation of anastomosing vessels can occur so that collateral circulation is gradually established, and the vessel may finally be completely closed without harm to the myocardium.

4. *Age.* — Anastomoses throughout tht coronary circulation including arteriae telae adiposae become more effective and more extensive with advancing years, so that, other factors being equal, the heart of a man between 50-60 is better prepared to withstand the effects of coronary occlusion than the heart of a man 20-25

Course of Events in Myocardium After Thrombosis.—In the vast majority of cases the pathological basis of cardiac infarction is endarteritis with atheroma of the coronary arteries. The final occlusion is due to thrombosis at the site of constriction of the vessel. Occasionally, cardiac infarction is due to closure of the mouths of the coronary arteries by syphilitic aortitis, with or without thrombosis in addition. The rarest cause of cardiac infarction is coronary embolism which is most often associated with an infective endocarditis.

The blocking of a main trunk of one coronary artery by a thrombus may cause sudden death, there being no myocardial change, since death occurs so suddenly there is no time for anatomical changes to take place. This is not always the case. When a branch of one of the coronary arteries is occluded, the blood supply is cut off from a circumscribed portion of myocardium and myomalacia cordis or an area of infarction results. The tissue in the affected area may undergo fatty degeneration, tending to rupture, or, if the area is quite small, muscle fibres may undergo necrosis with fibrous tissue replacement and complete repair is the final result. There is also the possibility of aneurysm formation because of the weakened, thinned-out wall. The aneurysm may enlarge and the thin wall rupture, giving rise to death.

(To be continued)

EMPYEMA OF THE PLEURA

By M. VERA PETERS, '34.

"Empyema of the Pleura," from the standpoint of the general practitioner, is the most frequent and the most important surgical disease within the chest.

Incidence.

Empyema is a disease of childhood particularly, although it may occur at any age. In adults it is much more common in males than in females, but in children there is an even distribution between the sexes.

Etiology.

The organism most frequently found in the pus is a pneumococcus. The streptococcus, however, is almost as frequent. Other less common organisms found are the staphylococcus and the tubercle bacillus.

Anatomical Factor.

Each lung is invested by an exceedingly delicate serous membrane, the pleura, which is arranged in the form of a closed invaginated sac. A portion of the serous membrane, the pulmonary pleura, covers the surface of the lung and dips into the fissures between its lobes. The rest of the membrane, the parietal pleura lines the inner surface of the chest wall, covers the diaphragm, and is reflected over the structures occupying the middle of the thorax. The two layers are continuous with one another around and below the root of the lung and in the healthy state they are actually in contact with one another, but the potential space is known as the pleural cavity. Thus the right and left pleura are entirely separate from one another, and between them lie all the thoracic viscera except the lungs These viscera constitute the mediastinum. The arteries of the pleura are derived from the intercostal, internal mammary, musculophrenic, thymic, pericardiac and bronchial vessels, the veins corresponding. The nerves are derived from the phrenic and sympathetic.

Physiological Factors.

Normally, the act of respiration depends chiefly upon an alternate expansion and contraction of the chest. During inspiration the enlargement of the thorax necessitates the inrush of air drawn into the lungs to equalize the pressure within and without the lungs. On expiration, the opposite occurs. Thus, during inspiration, the pressure within the pleural cavity is less than that of the atmosphere, and is commonly expressed as a "negative pressure" existing within the pleural cavity, which pressure has its lowest value at the end of inspiration It rises with the expiratory act, but under normal conditions it does not reach that of the atmosphere even at the end of expiration, unless the glottis is closed. The average intrapleural pressure at the height of inspiration is —4.64 mm. Hg., and at the end of expiration is —3.02 mm. relatively.

Therefore, respiration cannot go on unless at some time during the act of inspiration the intra-pleural pressure becomes less than that of the atmosphere.

Pathogenesis.

There are six ways in which empyema may develop.

1. The virulent organisms might enter the trachea and spread by direct extension by way of the alveoli and the visceral pleura. This mode of origin presupposes the occurrence of pneumonia of lobar variety and is probably the way in which most of the pneumococcal empyema originate.

2. If the virulent bacteria gain access to the peribronchial tissues they might spread directly to the pleura

through the interstitial lymphatics and tissue spaces. This presupposes an interstitial or peribronchial type of pneumonia, which in this case would be lobular or broncho-pneumonia, which is commonly associated with infections by the hemolytic streptococci. In an empyema associated with this type of pneumonia one would expect to find streptococci in the pus.

3. Infection by way of the blood stream is possible, but rare.

4. Empyema is sometimes caused by rupture of a lung into the pleural cavity. In this case, the more uncommon etiological organisms may be found, such as the staphylococcus and putrefactive bacteria of various kinds. Sometimes in ordinary cases of haemolytic streptococcal empyema, small abscesses are found immediately beneath the visceral pleura.

5. Occasionally empyema is the result of rupture of an abscess below the diaphragm into the pleural cavity, but this is very rare.

6. A common mode of infection during the war was by way of penetrating wounds.

Pathology.

Empyema constitutes an abscess of the pleural cavity. The amount of purulent exudate may vary from a few cubic centimetres to several litres, since it is governed by the character of the inflammation. Large exudates are more common in streptococcal empyema than the pneumococcal type. This is to be expected, however, since in the peribronchial type of pneumonia the pleura is liable to be attacked by the bacteria in several areas, whereas the lobar type of pneumonia confines the process to one lobe generally.

The pleural effusions in the early stages, or the formative stage, of an empyema may be serofibrinous, blood tinged or distinctly haemorrhagic. On removal these fluids tend to separate into layers, a layer of pus collecting at the bottom, the supernatant layer of fluid being serohaemorrhagic. The leukocytes found are mostly necrotic. The fluid often has a greenish cast. Throughout it are flakes and strands of fibrin. Later it assumes a thicker, more purulent character. If it is secondarily infected it becomes foul and often blackish green.

The visceral and parietal pleura become enormously thickened due to the *deposition of fibrin.* The parietal pleura may at times be 2 or 3 cm. in thickness. The fibrinous adhesions which are present in the early stages later become organized into fibrous adhesions. In the streptococcal cases *multiple* pockets of pus may be found.

In the pneumococcal type of empyema, the pneumonia is usually in the stage of resolution before the pus forms in the pleural cavity. In marked contrast to this, in the streptococcal type pus may be present in the pleura at a very early stage of the pulmonary infection. In the streptococcal pneumonia a peribronchiolitis exists with patches of pneumonia which may coalesce to suggest lobar distribution. The lumina of the bronchioles may be plugged with masses of necrotic material and leukocytes. Thus, a patchy atelectasis is common, but it may be so diffuse as to involve the whole lobe. In the streptococcal cases also, abscesses of the lung are common and may be confused with the interlobar pockets of pus which are not uncommon.

In chronic empyema the pleura becomes enormously thickened. In tuberculous empyema the reparative processes are not so pronounced as in pyogenic types of inflammation. Thus the pleura does not become so markedly thickened.

Signs and Symptoms And Diagnosis.

Fever.—Following the course of a typical case of lobar pneumonia, after the fever has come down to normal or nearly so, a sudden rise in temperature occurs and, if this high temperature persists, it is almost definite presumptive evidence that an empyema of the pleural cavity exists. This sudden fever

symptom may indicate unresolved pneumonia, which is rare and should not be diagnosed until empyema can be excluded. This observation applies also to any inflammatory condition of the lung. Any sudden rise of temperature and increased prostration of the patient is almost always evidence of extension of the pneumonic process or the beginning of an empyema. The diagnosis of empyema is made certain only by ultimate exploration, but the following symptoms may or may not be present:

1. Pain, rarely except at beginning of dry pleurisy.
2. Local tenderness.
3. Leukocytosis.
4. Sweating.
5. Chills.

The last three may occur at the beginning of an empyema due to rupture of a pulmonary abscess into the pleural cavity. This last condition may be spoken of as a pyopneumothorax and air as well as pus escapes into the pleural cavity. The presence of the air allows the fluid to take a level, to become more apparent, since it is now represented as a sharply demarcated line

Pyopneumothorax does not necessarily indicate the rupture of a pulmonary abscess, as it may be associated with an empyema caused by gas producing organisms.

The physical signs in an early acute empyema are very much the same as an ordinary pleural effusion. A great deal of information may be gained by inspection.

Inspection.

1. *Bulging* or fulness of the affected side. In children especially the disproportion between the two sides may be very marked.

2. *The intercostal spaces* may not only be obliterated, but may bulge.

3. There is frequently redness and *oedema of the chest wall,* but this usually indicates the beginning of an "empyema necessitatis" or spontaneous rupture through the chest wall.

4. Some degree of diminished movement is always present on the affected side depending upon the amount of fluid present.

5. The heart impulse may be displaced to the opposite side.

6. The network of subcutaneous veins may be very distinct.

7. Liver or spleen may be displaced downwards.

8. Occasionally there is pulsation of the side of the chest containing the empyema due to the transmission of the impulse from the heart and aorta through the fluid.

Palpation:—enables a more correct determination of:

1. The obliteration of the intercostal spaces.

.2 The degree of immobility of the affected side.

3. The position of the heart impulse.

Besides,—

4. Tactile fremitus is almost invariably diminished or absent in contrast to consolidation, where it is increased.

On Percussion the most exact information is gained.

1. There is flatness shading to dulness over the pus filled cavity so that a pretty accurate estimation of the outline and form of the empyema is gained.

2. There is also a peculiar feeling of resistance or wooden feeling transmitted to the percussing finger.

3. Above the fluid level the note is often tympanitic. ("Skodaic Resonance.")

4. The level of dulness changes very little with the position of the patient.

5. Graeco's sign of paravertebral dullness on the side opposite to the empyema may be present.

On auscultation.

1. Usually the breath sounds are very weak or entirely absent over the fluid, but occasionally, in children especially, bronchial breathing may be heard over the whole dull area. This latter finding may lead to a mistaken diagnosis of consolidation rather than fluid. However, diminished tactile fremitus distinguishes

fluid from consolidation, where it is nearly always increased.

2. Vocal resonance is diminished or absent over the fluid.

3. Whispered voice sounds are absent in frank empyema, but are only diminished in intensity in pleural effusion in contrast with an increase in their intensity in consolidation. This is not always true and there may be bronchophony.

4. Egophony is often present at the upper level of the fluid.

The *X-ray* pictures are very valuable in diagnosis. Plates should be taken in both antero-posterior and full lateral positions. They will reveal a clearly marked area of density. They will also demonstrate any spinal deviation; the contracted intercostal spaces and the displacement of the heart and other mediastinal organs to the opposite side. Where the effusion does not occupy the parietal pleural cavity, but is encysted either between the two lobes or elsewhere, the diagnosis is especially difficult since these cases are likely to be confused with lung abscess. Added to the X-ray findings, most of the later cases are associated with some degree of pulmonary suppuration and in many there is a direct communication with the lung resulting in the coughing up of varying amounts of purulent sputum. Where the interlobar empyema is advanced it shows up in X-ray as a band of density corresponding in general with the position of an interlobar fissure. But in lung abscess the shadow does not usually extend across the lung with such a definitely horizontal base. Other unsuspected conditions may also be discovered by X-ray, such as sacculated empyema or tuberculous disease of the lung.

The constitutional disturbances are similar to those of any severe febrile condition. Anorexia, emaciation, weakness and sweating may be conspicuous. The urine shows a small amount of albumen and a few casts may be present. The casts usually disappear after adequate drainage has been established, but in chronic empyema the damage to the kidney may be permanent.

The *temperature* of the patient varies a great deal during the course of the empyemic process. In the acute cases the temperature may be very high, especially in children, but in chronic empyema it may be practically normal. Temperatures above 104° F. usually indicate either a co-existing pneumonia or a complicating infection. The fever is usually of the septic type with small remissions.

The pulse rate is accelerated.

The blood count almost always shows some leukocytosis.

A secondary anaemia nearly always exists.

The vital capacity of the lungs is diminished.

Unless the proper nutrition is stressed the patient loses weight, sometimes quite rapidly. Experiments on the metabolism of patients with empyema have been carried out in which it was shown that with an average caloric intake of 15 - 1700 cal. per day the patients had a negative nitrogen balance amounting to a deficit of as much as 21 grams per day and this is not by any means due only to loss of nitrogen by way of pus which has drained each day.

Clubbing of fingers and toes, the pulmonary osteo-arthropathy of Marie is commonly seen in chronic cases of empyema. When the infection clears up the clubbing diminishes and may disappear.

Scoliosis of varying degrees is especially common in children, particularly in neglected empyema.

Complications.

The following are the most common *complications* of empyema, besides chronicity.

1. *Association with a Bronchial Fistula.*—This may be due to one of two processes. Either the communication has been made by rupture of an empyema into the lung, or by rupture of a pulmonary abscess into the pleura. In the latter case the empyema has been

caused by the rupture of a pulmonary abscess into the pleura. It has been found that frequently the pulmonary abscess clears up shortly after the creation of adequate drainage of the complicating empyema. But, on the other hand, the expectoration of pus alone will seldom give adequate drainage to allow satisfactory healing of the empyema.

2. *Empyema Necessitatis*—or rupture of the empyema through the thoracic wall. This takes place only after several months of neglected empyema. The abscess on the thoracic wall just before perforation presents all the characteristics of an abscess anywhere—redness, swelling, pain, tenderness, and fluctuation. In addition it gives an impulse on coughing and on forced expiration. The course of the sinus communicating the pleural cavity with the external thoracic abscess may be so tortuous that the perforation or the abscess may be far removed from the opening through the pleura. The spontaneous perforation does not usually afford adequate drainage for the healing of the empyema and operation is usually necessary. Osteomyelitis of a rib frequently complicates empyema necessitatis. Pneumococcal empyemas perforate less commonly than those due to other organisms. Actinomycotic infections perforate readily, thus simple abscesses of the chest wall should always make one suspicious of a pointing empyema.

3. *Suppurative Pericarditis* — is a common complication of streptococcal empyema usually. It is assumed that the infection is due to direct extension of the organisms through the pleura to the pericardium.

4. *Peritonitis*—is more common as a complication of the streptococcal variety of empyema than pneumococcal. It is usually generalized, but sometimes may be localized as a subphrenic abscess. A common explanation of the mode of spread of the infection is by direct extension through the aerolar tissue in the same manner as the spread of interstitial emphysema, but some are inclined to think of these complications as being blood borne, since they all seemed to be infections of serous membranes, the organisms in the blood bearing a special affinity to such membranes, causing a polyserositis. But blood cultures in most of the cases were sterile. Doubtless the infection passes through the diaphragm, presumably by way of the lymphatics, but it is more usual from below upwards than from above downwards.

5. *Blood Stream infection* occasionally takes place regardless of the organism, but it seems to be difficult to demonstrate positive blood cultures except just before death.

6. *Bilateral Empyema* is more common with streptococcal infections.

7. *Mediastinal abscess* is a common post-mortem finding, but is very difficult to recognize during life.

8. Other rare complications such as meningitis, brain abscess and multiple arthritis may occur.

Prognosis.

Empyema is a chronic affection and the majority of cases, if left alone, end in death. During the war the mortality of empyema ranged between 30 and 70%, but since, however, with the better understanding of the rationale of treatment, the mortality has dropped.

In general, pneumococcal empyema has a better prognosis than the streptococcal variety. Also the age of the patient has a marked influence. Empyema in babies under two years has a high mortality rate. In infants it is nearly always a complication of pneumonia and as such is only part of the picture of pneumonia which alone is very serious in small children.

Principles of Treatment.

The most essential principle in treatment is never to operate in the primary or formative stage when the process is still only an exudative pleurisy and the fluid is clear. The danger here is that of pneumothorax. One should aspirate only and wait until the fibrin has formed a thick wall over the

visceral and parietal pleura. Aspiration here has the advantage of reducing the size of the cavity and allowing more fibrous adhesions to keep it reduced. One should aspirate every two or three days in the formative stage, especially if the organism is streptococcus. Open operation in primary stage is indicated only when the infection is mixed and one fears rupture into the lung. After a thick wall of fibrin has formed, aspiration is useless. It is important to operate on empyema before it becomes chronic, however, i.e., when it has existed less than six months, according to N. F. Shenstone.

When operation has been decided upon, which is after obvious pus has been obtained on aspiration and after the pneumonia has practically resolved, the type of operation is then considered. In children aspiration alone may be sufficient. If the patient is in the hospital within convenient distance to the doctor, closed drainage is advised, since it does not permit the entrance of air or other organisms. If the patient is in the country, open drainage is advisable.

The object of drainage is the sterilization and obliteration of the pus cavity. Drainage should be carried on at least five weeks and usually longer. A case of empyema cannot be considered as healed until the cavity is completely obliterated. When the cavity is obliterated the lung is found firmly to the chest wall by adhesions, covering the entire extent of the old cavity. Repeated X-ray examinations will demonstrate the gradual disappearance of the cavity usually, but one of considerable size may be overlooked. The only certain method of estimating its size is freuent measurements of its fluid capacity. The removal of the drainage tube is not safe until the cavity measures less than ten cubic centimetres.

Chronic Empyema.

The term chronic empyema is usually applied to cases in which the process persists longer than about six months. This chronic empyema is much more common in adults. It is found either as a previously unrecognized accumulation of pus in the chest, or, more commonly, as a case presenting a discharging sinus. Often a patient may go a year or more with quite a large empyema without sufficient distress to warrant medical consultation. The only symptoms complained of may be a cough, pain in the side or loss of strength. Examination, however, reveals as well as the local physical findings, emaciation, more or less, which even may be extreme. Fever leukocytosis and scoliosis may be present. X-ray examination confirms the diagnosis.

The different types of discharging sinuses are:

1. Those leading from a relatively large cavity to the outside of the chest.
2. Those which communicate with the lung or with a bronchus, but do not lead into a large cavity.
3. Combination of the above two.
4. Sinuses which lead only down to the pleura or to a rib.

The most important causes of persistent sinuses are:

1. Inadequate drainage.
2. Presence of foreign bodies.
3. Communication with the lung.
4. Tuberculous or similar infections.
5. Failure of the cavities to obliterate themselves spontaneously.
6. Osteomyelitis of a rib before or after healing of the empyema cavity.

Inadequate drainage using the closed method may be due to plugging of the drainage tube, or the use of too small a drainage tube for a comparatively huge cavity. Again so much faith has been placed in suction that in many cases there has been no attempt to make the drainage opening at a dependent part of the cavity.

The foreign bodies which commonly produce chronicity are sequestra of ribs and pieces of drainage material such as tubing or gauze.

Communications with the lung are of two kinds,—

1. *Pleural pulmonary fistula,* possibly due in most cases to rupture of a subpleural pulmonary abscess. These usually heal spontaneously.

2. *Bronchial fistula*—where the empyema communicates with a large bronchus. This type is less likely to heal spontaneously and the fistula may become lined with epithelium. Bronchial fistulae may communicate with the outside and no other cavity exist.

Pleurisy due to tuberculosis and other similar types of infection such as actinomycosis are characterized by their lack of healing qualities.

Failure of the cavities to obliterate themselves spontaneously is dependent on two chief factors: —

(a) Fibrosis and atelectasis of the lung behind the cavity preventing expansion.

(b) Intensely thickened inelastic coat of exudate covering the exposed surface of the lung limiting its inflation.

Diagnosis of Chronic Empyema.

The *diagnosis* of chronic empyema is made on the history, physical findings and X-ray examination. The patient usually tells of an influenza or pneumonia which did not clear up entirely or they may give a story of rheumatism or persistent pain of a dull character, or "Cold with persistent cough." These patients often feel ill and feverish at times, but continue to attend to their duties.

The patient usually appears chronically ill, undernourished and pale-faced, with a dull facial expression. In addition, clubbing of the fingers and toes, slight temperature, leukocytosis, as well as the local findings may be present. X-ray examination reveals an opacity of the affected side of the chest which may extend over the entire half of the chest or which may be very irregular in contour, as well as dislocation of the mediastinal contents. Diagnostic aspiration will reveal the presence of pus. Smears and cultures should be made from the secretions as well as any persistent sinus.

If the cavity is suspected of being multilobular, the size and contour of the cavity may be discovered by various methods, such as instrumental exploration with a long probe, such as a uterine sound, or by X-ray. The injection of lipiodol before X-ray will reveal not only the size and contour, but any communication with the lung. Usually in order to rule out the question of the presence of a foreign body in a case of chronic empyema it is necessary to operate in order to obtain a satisfactory exposure of the cavity.

Treatment of Chronic Empyema.

The chief *principle of treatment* is to provide adequate drainage, since the majority of chronic cases are due in part to inadequate drainage. This can be done generally by the resection of one or two ribs. Proper inspection of the cavity may be made in this way. If the parietal pleura is not greatly thickened there should be a strong suspicion of tuberculosis, and it is always well to remove a section for microscopic examination.

After the creation of adequate drainage, the irrigation of the cavity with Dakin's solution is the next procedure. Any more radical operation should be postponed until the above procedure determines whether or not the cavity will obliterate itself. Astonishingly large cavities have been reduced in this way within six or eight weeks. Finally, if the above efforts have failed to reduce the cavity, it will be necessary to remove the roof of the cavity, and further resect the overlying ribs and thickened parietal pleura. The obliteration of the cavity is also greatly assisted by decortication of the thickened visceral pleura in order to permit greater expansion of the lung.

Tuberculous Empyema.

Tuberculous involvement of the pleura may be either primary or se-

condary to pulmonary tuberculosis. When the character of the exudate becomes definitely purulent a tuberculous empyema exists. The organisms from the pus in smears and cultures are difficult to detect, but innoculation into guinea pigs is usually successful in detecting the tuberculous bacillus. The chief distinguishing factors between an ordinary pyogenic empyema and a tuberculous empyema are:

1. Under adequate drainage the tuberculous patient continues to be septic with high fever, rapid pulse, etc.

2. The parietal pleura is not as markedly thickened.

Many patients with a mixed tuberculous and pyogenic infection have been put into their distress as a result of unwise treatment by the establishment of open drainage on a case of serofibrinous fluid in the pleural cavity.

If repeated aspirations and ordinary hygienic treatment fail to obliterate a simple, uncomplicated tuberculous empyemic cavity, one should resort to the operation of thoracoplasty. Tuberculous empyema by this treatment has been converted from a condition with a mortality of almost 100% to one with an almost negligible mortality.

In conclusion I shall quote Evarts A. Graham's summary of the essential points in the principles of treatment of empyema of the thorax.

"(1) Careful avoidance of open pneumo-thorax in the acute stage.

(2) The prevention of a chronic empyema by the rapid sterilization and obliteration of the infected cavity, and

(3) Careful attention to the nutrition of the patient."

WRITTEN BY A JAPANESE STUDENT.

To Hon. Pepys, who edit Tonics with Sedatives with gusto:

Dr. Editor, I am Japanese student of medicine with difficulty. My cousin Ito ask me, Togo, are cancer hereditary, like barrenness in women, or do it merely run in family? I tell my cousin I look up matter in Journal of Cancer Research and let him know.

Dr. Editor, I know less now than before. Hon. Maude Slye say cancer in her mouses are mendoolian regressing. Fine! I should stop reading here, then I know something. But I read further, and Hon. Little, Pres. of Ann Arbor Mich, say she put too many red stars in reports to mislead casual reader. Hon. Slye renig. that this are not casual reading matter in the first place and those red stars are nothing to what she could put if she feel like. She say cancer in mouse are not sex-limited, but predominating in female. Hon. Little say (I quote verbum sap): "It is sex-limited, it's somatic appearance being in female mice in 99.4% of recorded cases." Dr. Editor, if it are sex-limited how do they occur in 0.6% of male mice? Perhaps these 0.6% male mice not regular fellers? I ask to know.

Hon. Little state that nobody in world but Hon. Slye own any cancer-free breeding stock. He try everywhere to get some, but Hon. Slye say if you want cancer-free breeding stock send to Sears-Roebuck. I quote verbum sap again: "If Miss Slye doesn't want any of her stock to get out of lab. I will be very glad to pay the expenses of a person who will take a no. of pregnant female mice and breed them with Miss Slye's males, while she retains the male in her lab." He say this on p. 279 of Journal of Cancer Research for Sept., 1928.

Dr. Editor, if you breed preg. fem. mice what is going to happen anyway? I fear you get not only cancer to run in family, but preg. too.

Hoping you are the same, I close,

Hoshimina Togo.

CARCINOMA OF THE COLON*
(EXCLUSIVE OF THE RECTUM)

By E. M. LIMBERT, '34.

The cases of cancer of the large bowel which one sees in hospital have one outstanding point in common: they are all well advanced, most of them to a stage where operation holds only a small chance of permanent cure. It is with this in mind that I intend to lay the greatest stress on diagnosis in this paper.

The etiology has been discussed at length by various authors, but nothing has as yet been proven. Statistics suggest that it is more common in men than in women, yet a series of 96 cases in the Victoria General Hospital in Montreal shows an almost equal sex distribution. That heredity plays an important part is generally conceded. Predisposition of the tissue to malignant growth as a factor is shown by the comoparatively high (3%) incidence of polyposis in cases of colonic malignancy, but chronic ulcerative colitis is rarely an associated lesion. There appears to be an anatomical feature in that many of the growths are situated at points where there is a definite interruption of the wall, as the ileocaecal valve, the appendicular junction, the rectal folds, and the anus, and at points where the wall is more or less fixed so as to increase the effect of movement of the contents, as at the caecum, the flexures, the rectal valves, and the point of contact of the rectum and prostate. Similarly, the physiological hardening of the bowel content as it progresses is probably responsible for the increased frequency with which carcinomatous changes take place in the left colon. Most authors are agreed that the order of frequency in which the parts are attacked is as follows: sigmoid, splenic flexure, caecum, ascending colon, and transverse colon.

Pathology.

The detailed pathology is the subject of much interesting controversy, but for the purpose of this paper I must restrict myself to the two major types of growth. The growth which most frequently attacks the caecum, ascending colon, hepatic flexure, and the right half of the transverse colon is typically described as being a papillary adenocarcinoma. This forms a large fungating mass which projects into the lumen of the bowel, bearing glands which secrete much mucus, and exhibiting a tendency to early ulceration, and colloid degeneration. As might be expected, blood appears *mixed* with the stools early in the disease, and obstruction is a late feature due to the fluidity of the faeces at the site of the growth.

The left bowel, on the other hand, tends to be afflicted by an annular fibrocarcinoma, characterized by a large amount of fibrous tissue in proportion to the amount of malignant tissue present. The growth undergoes a cicatricial contracture resembling that occurring in fibroadenomata of the breast, so that the lumen is progressively reduced. Owing to the hardness of the faeces at the commoner sites of these growths the blood is on the outside of the stool, and obstruction is often the first feature to attract attention. Cancer of the large bowel is commonly single, and metastasis to the liver or the regional lymph nodes occurs late in the disease.

The presence of the growth sooner or later results in partial obstruction, which is compensated for by dilation and hypertrophy of the bowel proximal

*Read before the Hippocratic Society, Nov. 30, 1933, in conjunction with a paper on Carcinoma of the Rectum by A. B. Noble, 3.

to the growth. The irritation of the inspissated faeces may give rise to stercoral ulcers which produce pus and in some cases perforate into the peritoneal cavity or an adjacent viscus, the ulcers being in the growth or situated some distance above it. Infiltration and contraction of the surrounding mesentery are late events. Complete obstruction may occur at any time due to stenosis, tumor bulk, adhesions, or contraction of the mesentery, or any of these plus unusually solid faeces such as those following a barium meal.

Diagnosis.

The symptoms as described in the text-books usually begin with "alternate constipation and diarrhoea," and the milder changes preceding this, when it occurs, are assumed by many of us to be due to chronic constipation and given scant attention. In the early stages the growth is often silent, or at most produces changes which are only brought out by painstaking questioning of the patient regarding his bowel habits. Thus in the taking of routine history of a patient who comes with some apparently unrelated complaint of gastric distress, one discovers that in the last two or three weeks he has required a laxative twice a week in order to produce a daily evacuation, whereas previous to this his weekly or bi-monthly opening medicine was a habit rather than a physiological necessity. This stage of the disease may easily be missed by the physician and accredited by the patient to lack of sufficient roughage, yeast, oil, or other widely-advertised aid to intestinal regularity. Closer attention to the intestinal function may disclose the recent occurrence of reflex gastric phenomena, such as slight loss of appetite, a sense of delay in emptying of the stomach or heaviness or other discomfort following meals, or gas and flatulent distension unusually persistent in the individual patient under consideration. In other patients the chief complaint may at once attract attention to the colon. Consciousness of increased intestinal activity in the lower abdomen and disturbing borborygmi may be followed after a time by marked constipation. A mild attack of colic occurs and is relieved by a profuse discharge of faeces and mucus. If the patient has taken a laxative the diarrhoea and even the colic pass as phenomena to be expected. Some will seek medical aid after one such attack, some will have a history extending back over many weeks, and some will try home remedies until acute obstruction forces them to seek relief elsewhere. There are a few unfortunate ones in whom the last-mentioned is the first danger signal.

In some cases bleeding occupies a prominent place in the history, and rightly so. Any case of bleeding from the bowel should be investigated until the presence of cancer is either proven or as definitely disproven as modern methods will permit. And any person who complains of recent disturbance of bowel habit resembling those described in the preceding paragraph should have their faeces examined for blood in the manner to be detailed later. In some cases it has been known to pass unnoticed until the occurrence of a profound secondary anaemia. To complete the symptomatology, pain other than that due to colic or acute obstruction occurs in some cases as a late event. It may be due to the infiltration of the nerves or adjacent structures by the growth, the spread of secondary infection, or a chronic perforation similar to that occurring in duodenal ulcer, and is generally referred to the lower part of the back. In these cases in which such pain is the reason for medical consultation, other evidences of its cause will almost certainly make themselves evident. Recent loss of weight arouses immediate suspicion, and cachexia is of grave prognostic significance.

Examination of the abdomen in the most favorable cases reveals nothing, or at most an indefinite mass. The mass may be completely removed by a dose of castor oil, or the purging may

remove the flatulent distension, permitting accurate outlining of the tumor. Advancing obstruction produces first a local tympanitic enlargement of the lower abdomen which is later masked by the generalised distension of complete obstruction, accompanied by obstipation and succeeded by intermittent attacks of crampy pain and vomiting. The crampy nature of the pain is often more evident from watching the patient than from the answers to questions which are purposely general in nature.

In preparing the patient for rectal examination, a procedure which is too often omitted from a mistaken sense of delicacy, traces of blood may be seen on the linen and about the anal orifice. Bleeding hemorrhoids may be seen or felt, and, especially if of recent occurrence, should be accepted as evidence of obstruction higher up until the contrary is proven. Growths on the region to which this paper is restricted may occasionally be felt, more frequently when the examination is aided by asking the patient to strain and by pressing the abdominal contents into the pelvis with the free hand. Particular attention should be paid to the presence of blood on the examining finger, and none to its absence.

In the presence of bleeding from the bowel the patient is certainly entitled to proctoscopic and sigmoidoscopic examination, valuable procedures which are much less specialized than the general attitude of the profession to them would seem to indicate. Proper preparation in the form of purging, a fluid diet for twenty-four hours, and enemata until the bowel is cleared three hours before the examination, is essential. The obturator is used until the internal sphincter is passed, and the instrument then slowly advanced with air insufflation under the guidance of hand and eye. Suspicious areas should be wiped free of mucus to render their features more distinct, and if a mass is seen or felt, a portion of it should be excised for closer examination, preferably using a cautery loop to avoid dissemination of metastases. In the absence of definite evidence of malignancy when no blood is seen coming down from beyond the farthest point reached by the instrument, a visible source of blood may be shafely assumed to be the only source.

Some writers lay great stress on the shape of the stools, but most ribbon-stools are the result of a tight sphincter acting an plastic intestinal content. Gross blood is unnoticed by many patients, and they should be instructed to watch for it over a period of several days. The tests for occult blood are only of value when the sample follows three or four days of meat-free diet. Pus visible to the eye is evidence of ulceration which may or may not be milignant, and demands miscroscopic exemination to explain its presence. One may occasionally find decreased or absent gastric acidity accompanying colonic carcinomata, according to some, but no adequate explanation has as yet been offered.

Roentgenoscopic examination of the colon is a valuable but expensive tool in the diagnosis of large bowel lesions, and though I do not feel justified in going into details here a few points are essential. A barium meal should never be given if any obstruction is suspected, as the resulting cement may precipitate acute obstruction. In any case the enema will serve the same purpose when the history and other findings are suggestive of a lower bowel lesion. When the results are negative a repeat is often advisable, and observation after evacuation of the enema and after air insufflation may bring out a lesion which is hidden when the bowel is full of barium. Diverticulitis and multiple polyposis are the commonest sources of error here. The former usually presents a long history of intermittent attacks of "right-sided appendicitis" and the mass as visualised is several inches in length. The latter is characterizzed by painless bleeding and a typical picture when the bowel, lined with barium,

is ballooned with air. If the diagnosis is still in doubt and the symptoms justify it, an exploratory laparotomy will make it a clear, as well as enabling the surgeon to make an estimate as to the size of the growth and its probable termination.

Treatment.

Having made the diagnosis, the course of treatment to be followed is to be determined by the features of the individual case. Those cases which are neglected until acute obstruction has occurred leave but one course open. The obstruction must be treated at once by some simple method, leaving the primary less-urgent condition until the patient is in good enough general health to stand the severe shock of extensive surgical manipulation, and the best method of doing this is to perform a caecostomy, a comparatively minor operation which may be done under local anaesthesia. The pouch of caecum may be safely opened in twenty-four hours with a cautery, and the obstruction relieved. Subsequent treatment is the same as in the cases which have been detected earlier, and is based on the results of a thorough investigation, particularly in regard to the general condition of the patient, the site of the growth, the degree of obstruction present, the presence of demonstrable secondaries, and the extent of infiltration of the surrounding tissues. Some of these factors are best evaluated at operation.

The detailed technique of the many operations employed is covered by standard works on the subject, but the principles apply to all of them. An attempt to minimise shock is desirable, and those cases which have had a caecostomy should be recovered sufficiently to have been out of bed for a few days. If conditions permit, the growth may be removed in one stage by Mickulicz' or Paul's method, or a wide resection with anastomosis done. A two-stage operation may be necessary, and in these the first stage is limited to the making of a permanent or temporary colostomy above the growth, to be followed when complete recovery has occurred by ablation of the diseased area. Cases which are hopeless from the standpoint of permanent cure may have their fatal termination deferred and their exit rendered less painful by simply relieving the obstruction.

From all aspects carcinoma of the large bowel is a most discouraging disease. Its presence is unsuspected in fifty per cent. of the cases until gross loss of blood has occurred, and a large number first demand attention when acute obstruction has supervened, though the history of bowel disturbances extends back over many months. The operative mortality is about eighteen per cent., an insignificant figure when one discovers that only one-fifth of all cases operated upon survive for a period exceeding ten years. The high mortality serves to make more clear the point with which this paper began: the importance of paying attention to minor disturbances of bowel function.

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CARCINOMA OF THE RECTUM*

By A. B. NOBLE, '34.

Carcinoma of the rectum is a subject of vital importance to the members of the medical profession. As students we cannot help but be impressed with the fact that many of the patients that we see with this condition are advanced cases at the time of admission. We also realize that the future welfare of any patient suffering from this condition depends upon early diagnosis. It is one of the commonest sites of cancer. The death rate from cancer of the rectum in the State of Massachusetts averages almost 400 a year. Dr. Daniel Jones of Boston states that in his experience the average case is not diagnosed until eight months after the onset of symptoms. One reason for this is that the early symptoms do not send the patient to a doctor. Most of these patients are elderly. Many of them suffer from chronic constipation. Many of them think they do. They treat themselves accordingly. They explain any change in the character of the bowel movement as being due to the effect of the laxative which they are using at that time. Many others must attribute the changes to haemorrhoids, and let it go at that. The other reason is that the doctor does not make an early diagnosis when he is consulted because he does not get a thorough, meticulous history.

On being asked to discuss the symptomatology of cancer of the rectum, there are several things we would think of. There is increasing constipation —many books mention alternating constipation and diarrhoea—also haemorrhage, tenesmus, mucus, ribbon stools; one would also expect to find anaemia and loss of weight. The alternating constipation and diarrhoea and ribbon stools mean stenosis; the blood and slime mean ulceration. On rectal examination of such a case we would find an ulcerating, annular constriction with probable massive involvement of surrounding structures. The condition has been there for many months, and the patient will die. In these late cases there is only one step, and that is to prevent a large bowel obstruction by means of permanent colostomy. The diagnosis in these cases is made too late. How, then, is this condition to be diagnosed in the early stages? There seems to be only one rule to follow in these cases. Dr. D. F. Jones of Boston, in an article appearing in the Canadian Medical Journal of May, 1933, states that every case showing symptoms due to any change in bowel habits or sensations -- and bleeding — must be considered to be due to malignant disease in the rectum until proven otherwise.

Pathology.

The adeno-carcinoma is the only type met with in the rectum. There are three clinical varieties, differing in physical characteristics and in degree of malignancy. These are: (1) the papilliferous carcinoma; (2) the common adenoid carcinoma; and (3) the colloid carcinoma.

The papilliferous type is an exuberant growth tending more to surface proliferation than to extensive invasion of the muscularis mucosa. This type of tumor is characterized by rapid surface extension and proliferation, causing obstruction of the bowel before there is any marked muscular involvement. As a result, extra-mural metastases do not form until the growth has been active for some considerable time. The surface usually begins to slough early, thus partially destroying the papilliferous character of the tu-

* A paper presented to the Hippocratic Society, December, 1933, in conjunction with the foregoing paper on Cancer of the Colon by E. M. Limbert.

mor. The retro-rectal lymph glands are often enlarged, more often due to septic absorption than to the formation of metastases. These tumors are usually recognized before extensive dissemination has occurred.

The majority of carcinomas of the rectum are of the adenoid type. The surface is flattened and the tumor rapidly extends in all directions, soon becoming adherent to and invading the musculâris mucosa. As the growth proceeds the surface sloughs and a definite ulcer with nodular, everted and indurated edges, is formed. Extension of the ulcer causes stenosis of the bowel with symptoms of impending obstruction. This type of tumor metastasizes very early. Wide dissemination has usually occurred before the onset of severe symptoms.

The colloid type is characterized by mucinoid degeneration of the epithelial and connective-tissue elements. They are highly malignant but are comparatively rare.

It would be well at this time to consider the various ways in which cancer of the rectum may spread.

First, there is extension through continuity of tissue. This occurs by gradual growth along the mucosal surface and by penetration of the musculature of the wall. The surface growth is more rapid, the growth having a tendency to grow more rapidly in the transverse than in the longitudinal direction. The invasion of the muscle wall, and later of the peri-rectal fat and the facia propria, is a slow process. Direct extension can then continue to surrounding structures — sacrum, prostate, bladder and vagina but according to Dr. Ernest Miles, these structures cannot be invaded for at least a year after the onset of objective symptoms, because of the slowness of direct extension in this manner. If this were the only mode of dissemination to be considered, many of these cases could be effectively cured by local removal of the diseased tissues.

There is a more or less theoretical mode of dissemination of malignant cells from the rectum, which, however, is seldom met with in actual practice. It is possible for dissemination to occur into the haemorrhoidal veins and for the cells to be carried through the portal system to the liver.

By far the most important mode of spread, and the one that is carefully investigated by every surgeon, is the involvement of the lymphatics. The lymphatic drainage of the rectum and anal canal occurs in a downward, lateral and upward direction. The vessels of the anal canal run with the inferior haemorrhoidal artery and vein across the ischio-rectal fossa, and communicate with internal pudendal vessels in Alcock's canal. These in turn open into the internal iliac lymph glands. From the lower part of the rectum drainage occurs into a lymphatic plexus situated on the Levator Ani muscles, which also communicate with the internal iliac glands. The lymphatic vessels of the upper part of the rectum accompany the superior haemorrhoidal vessels, and after traversing the para-rectal lymph glands, communicate with the lymphatics at the bifurcation of the common iliac artery. Efferents from the highest para-rectal group drain into those of the sigmoid mesocolon, which in turn communicate with the pre-aortic group at the origin of the inferior mesenteric artery.

The direction in which the cells travel is largely influenced by the direction of the current in the lymphatics which have been invaded. It must be remembered, however, that the path by which the cells advance is not entirely controlled by anatomical considerations. Thus it may happen that cancer cells derived from a growth in the rectum, wherever situated, may traverse the lymphatics in a downward, lateral or upward direction, or in all three directions simultaneously. During the transit their progress may be arrested at any point in the region traversed by the lymphatics and so lead to the formation of nodules. The tissues through

which the lymphatics pass are therefore prone to metastatic deposits, which may be either microscopic or macroscopic in character. It may be regarded as an axiom that whenever a visible metastasis exists, other metastases, which cannot be recognized without the aid of a microscope, also exist along the course of the lymphatics at points more distant from the seat of the primary growth. (Miles).

Symptoms and Signs.

The symptomatology of cancer of the rectum is certainly confusing. It is extremely difficult to mention a series of symptoms which can be ascribed to this condition without consideration of the various types of tumors and their most common sites within the rectum. Malignant disease is found in three situations: (1) at the recto-sigmoidal junction; (2) in the anal canal; and (3) at the ampulla.

These symptoms may be tabulated as follows:

(1) At the recto-sigmoid junction.
  (a) The papilliferous tumor.
    (i) Slight diarrhoea, which has a tendency to become more marked, due to increasing mucous secretion.
    (ii) Bleeding, which may become excessive if extensive necrosis occurs.
    (iii) Impending obstruction due to intussusception of bowel or impaction of faeces above the stenosis.
  (b) The adenoid tumor.
    (i) Rigid stenosis of the bowel, with signs of large bowel obstruction.
    (ii) Obstruction not preceded by diarrhoea or bleeding. The urgency of symptoms in these cases usually cause patients to seek advice early. As a result, many of these are operable.

(2) In the Anal Canal.
  (a) The papilliferous tumor.
    (i) Rectal irritation, sense of fullness and incomplete relief after defaecation.
    (ii) As superficial necrosis occurs there is pain on defaecation, bleeding and mucoid discharge.
  (b) The Adenoid Tumor.
    (i) Constipation, due to muscular inertia.
    (ii) Later, there is frequency. There is no pain, and often little or no mucus and blood. This is the common type that does not have distressing symptoms until late.

There are few significant points with regard to the ampulla. If the growth involves the lower part, there may be exquisite pain on defaecation, simulating the pain experienced in fissures of the anus.

The above-mentioned symptoms may merge almost imperceptibly with those mentioned in an earlier part of the discussion. Consideration of the above, however, will serve to impress upon our minds the fact that the symptoms or sequence of symptoms commonly associated with carcinoma of the rectum are indicative of more or less advanced disease. I think that most students, having had little practical experience with this subject, place considerable importance on the consistency of the stools, with special reference to the so-called "ribbon-stool." Lockhart-Mummery states emphatically that this symptom is of little or no importance. He maintains that the stools are formed by the last orifice through which they pass. In such a case we cannot expect this sign in cases where the tumor is situated anywhere but in the anal canal, and tumors in this situation are relatively rare.

Diagnosis.

We should all be acquainted with the various methods of examination employed in cases suspected of cancer of the rectum. As students, the important thing for us to acquire is the

ability to do a thorough digital examination.

Treatment.

The determination of the operability of a cancer of the rectum is another point which merits grave consideration. Not only must the surgeon decide as to whether or not the tumor is operable, but also the type of operation most suitable for the case in question. The age of the patient, the general condition of the patient, the situation of the growth, and the size of the growth and the time it has been present, must all be carefully considered. These points all demand the application of judgment acquired by long experience, and few general rules can be mentioned. P. Lockhart-Mummery advises against operation for rectal malignancy in patients over sixty-five years of age, as a general rule. The mere factor of age, of course, is of secondary importance to the general condition of the patient. The presence or absence of obesity, arterio-sclerosis, diabetes, a feebly-acting heart, or Bright's disease must, of course, be kept in mind, as in the case of any major surgical operation.

The situation of the growth is another matter of great importance. Growths within reach of the finger are much more favourable for removal than those at the upper end of the rectum. The surgeon also considers the condition of the growth locally, and attempts to discern as well as possible the amount of extra-mural involvement. If the diseased part is not mobile, and there is evidence of fixation to the sacrum, prostate, bladder, uterus, vagina or to the wall of the pelvis, the condition is generally considered to be inoperable. Such involvement by direct extension we know can only occur after a period of many months. From our previous remarks, we also appreciate that the tumor may still be mobile, and wide dissemination have taken place through the various lymphatic channels. Even after careful consideration of the above factors, there is no doubt that in many cases the chances of recurrence are very difficult to determine. In some types the tendency to return is marked; in others very slight. It is, on the whole, almost impossible to distinguish between these types of growths.

Generally speaking, there are three methods of operation commonly employed. There is the perineal operation, usually undertaken where there is a small growth situated in the lower part of the rectum or in the anal canal. Secondly, an abdomino-perineal resection is done in those cases that are so far advanced that extensive operation is necessary in order to eradicate the disease. Lastly, in the most advanced cases, where operative procedure is out of the question, a simple colostomy is done in order to prevent obstruction.

The most important object to be aimed at in any case where removal of the rectum is necessary, is aseptic healing. This cannot be achieved if faecal material be allowed to pass over the wound area after operation. In order to render the area aseptic, a preliminary colostomy is always done in order to deflect the bowel contents. This is usually done a week or ten days previous to operation. At the same time, an opportunity is afforded the surgeon to explore the abdomen and determine the extent of the metastatic involvement of the lymphatics and surrounding structures. The colostomy is opened two days later, and the lower bowel and rectum are then washed out daily from the lower colostomy opening.

The operative mortality in these cases shows wide variations. Lockhart-Mummery places his mortality in cases of perineal excision at approximately 3%. The operation is unfortunately of use only in selected cases, where the growth is low and where only the lowest group of lymphatic glands need be removed. The abdomino-perineal excision is a very formidable operation. The operative mortality — as describ-

ed by various authors — varies from 20% to 30%. This type of operation is practicable in only certain cases. People over sixty years of age cannot stand such a procedure unless their general condition is excellent.

Authorities agree that radium therapy has no place in the treatment of carcinoma of the rectum.

The after-treatment of these cases is extremely important. The posterior wound will not heal by first intention, and must therefore be watched very carefully and dressed twice a day. A new pelvic floor of peritoneum is made at operation, and it is often necessary to support this for the first few days after operation. Intravenous therapy is usually found to be very beneficial. Good nourishing food is allowed in generous quantities as soon as the patient desires it. The patient's strength is thus built up again as soon as possible. The patient is usually allowed up about fourteen days after operation, if the general condition warrants it. These people can often resume work after a period of a few months, providing a great deal of physical exertion is not required. The disability suffered by the colostomy opening is in many cases much less than the patient expects. A pouch forms in the terminal bowel, and the patient soon gets along very well by attending to the colostomy once every twenty-four hours. Several very satisfactory methods of irrigation have been devised. One such device consists of a metal cap which fits over the opening. It is fitted with a small rubber tube through which water is supplied. The products of irrigation are removed through a larger tube. The cleansing by this method requires about half an hour. It is thorough, and is not obnoxious to the patient. These patients are able to resume their place in society, and are often able to enjoy years of good health. Even if not cured, the last years of their life are made much more bearable.

References.

Jones, C.M.A.J., May, 1933.
Miles, Cancer of the Rectum.
Lockhart-Mummery, Diseases of the Rectum and Colon.
Gray's Anatomy.

In naturall philosophye
Thy studye shoulde be bente;
To knowe eche herbe, shrubbe, roote and tree,
Muste be thy good intente.

Eche beaste and foule, wyth worms and fishe,
And all that beareth lyfe;
Their vertues and their natures bothe,
With thee oughte to be rife.

And in the grounde metall and stone,
And veines of earthe also;
Their powres and vertues in degre,
Shoulde not be hid the fro.

But chieflye the anatomye,
Ye ought to understand;
If ye will cure well anye things,
That ye doe take in hande.

From "An Historiall Expostulation: against the beastlye abusers, bothe of Chyrurgerie, and Physyke, in oure time, with a goodlye Doctrine and Instruction, necessarye to be marked and followed, of all true Chirurgiens:

Gathered and diligently set forth by John Halle, Chyrurgyen. Imprinted at London in Flete Streate, nyghe unto Saint Dunstones Churche by Thomas Marshe. An. 1565."

BOOK REVIEWS

A MANUAL OF EMBRYOLOGY: J. E. Frazer, F.R.C.S., Eng. London: Bailliere, 1931.

That such a first-class book on Human Embryology should be practically unknown is a pity, for it is one of the very few books that deal with Human Embryology, fully and solely. Frazer does draw analogies and comparisons with the Chick and Salamander but he is always careful to interpret his findings in terms of the human development. The treatment of the gill arches is masterful. The section on the development of the stomach and intestine is based upon research carried out by Frazer for many years. By reading the book many reasons are found to explain the juxtaposition of the adult anatomical structures, and thus is furnished a background to anatomy that is indispensable for a thorough understanding of the principles of anatomy and their application to Medicine. There are many excellent sketches and pictures in the book. A grounding in embryology is necessary to fully understand and appreciate this admirable work.

DISEASES OF TRADESMEN: Bernardino Ramazzini: New York Medical Lay Press. 1933.

This little book by Ramazzini is written in the quaint scholarly style of about 1700 A.D., for it is written in that style peculiar to the universal language of the educated man of that period, namely, the concise, direct phraseology of the Latin language. But for all that, it has a grace and charm; a naive spirit almost amounting to flippancy that makes it delightful reading. Ramazzini was a well-known physician of the times, and the manner of his research shows a true love of the fundamental cause of things. His discussion at no time is profound, but it brings to mind the need for being familiar with every walk of life and every mode of livelihood in order to carry out the practice of medicine in its modern phase to the best of one's ability.

With this volume is bound Silk Handlers disease of the Skin by Herman Goodman.

It is a delightful book which is well suited to take its place among other historical writings on Medicine.

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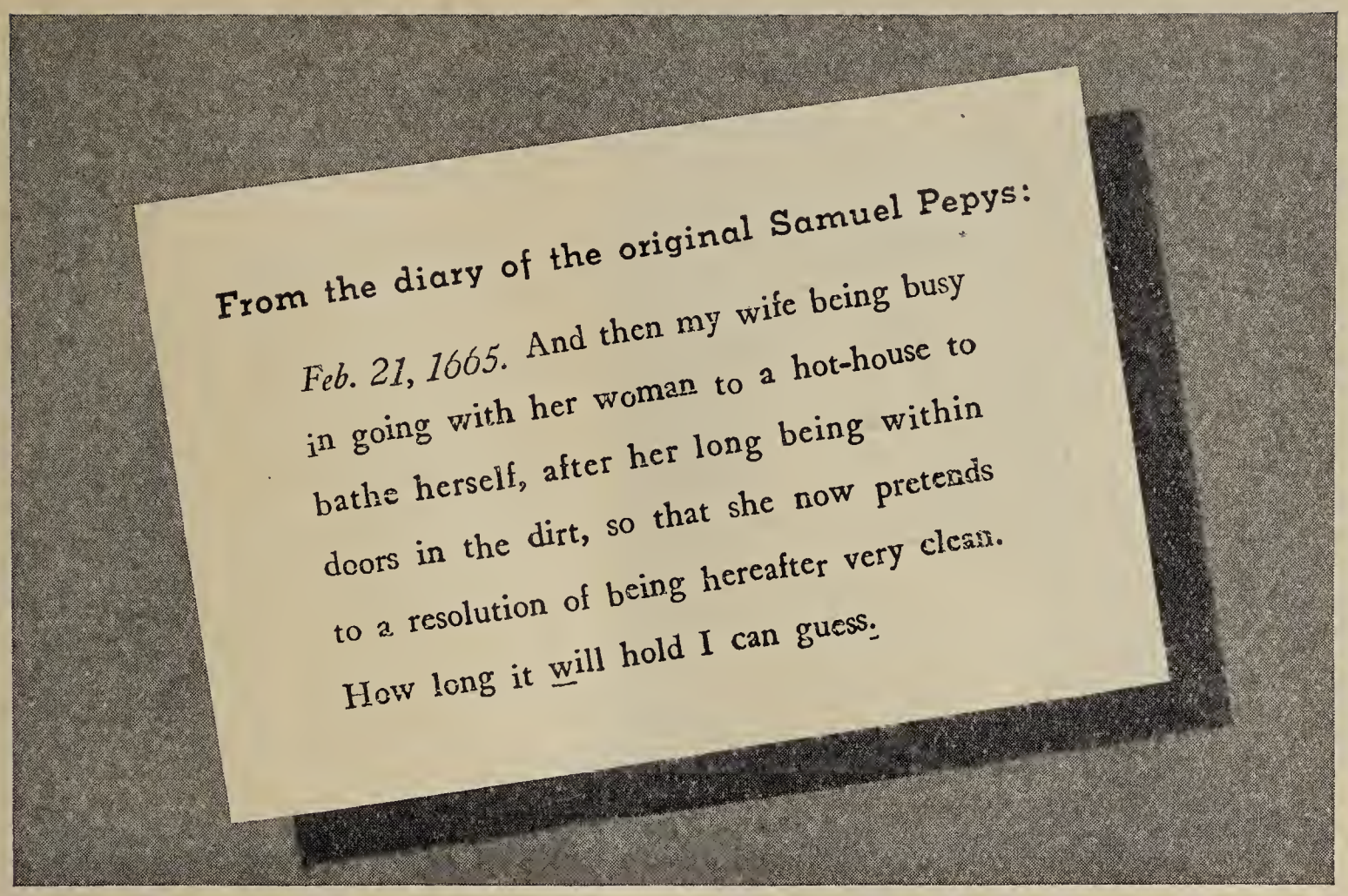
From the diary of the original Samuel Pepys:
Feb. 21, 1665. And then my wife being busy in going with her woman to a hot-house to bathe herself, after her long being within doors in the dirt, so that she now pretends to a resolution of being hereafter very clean. How long it will hold I can guess.

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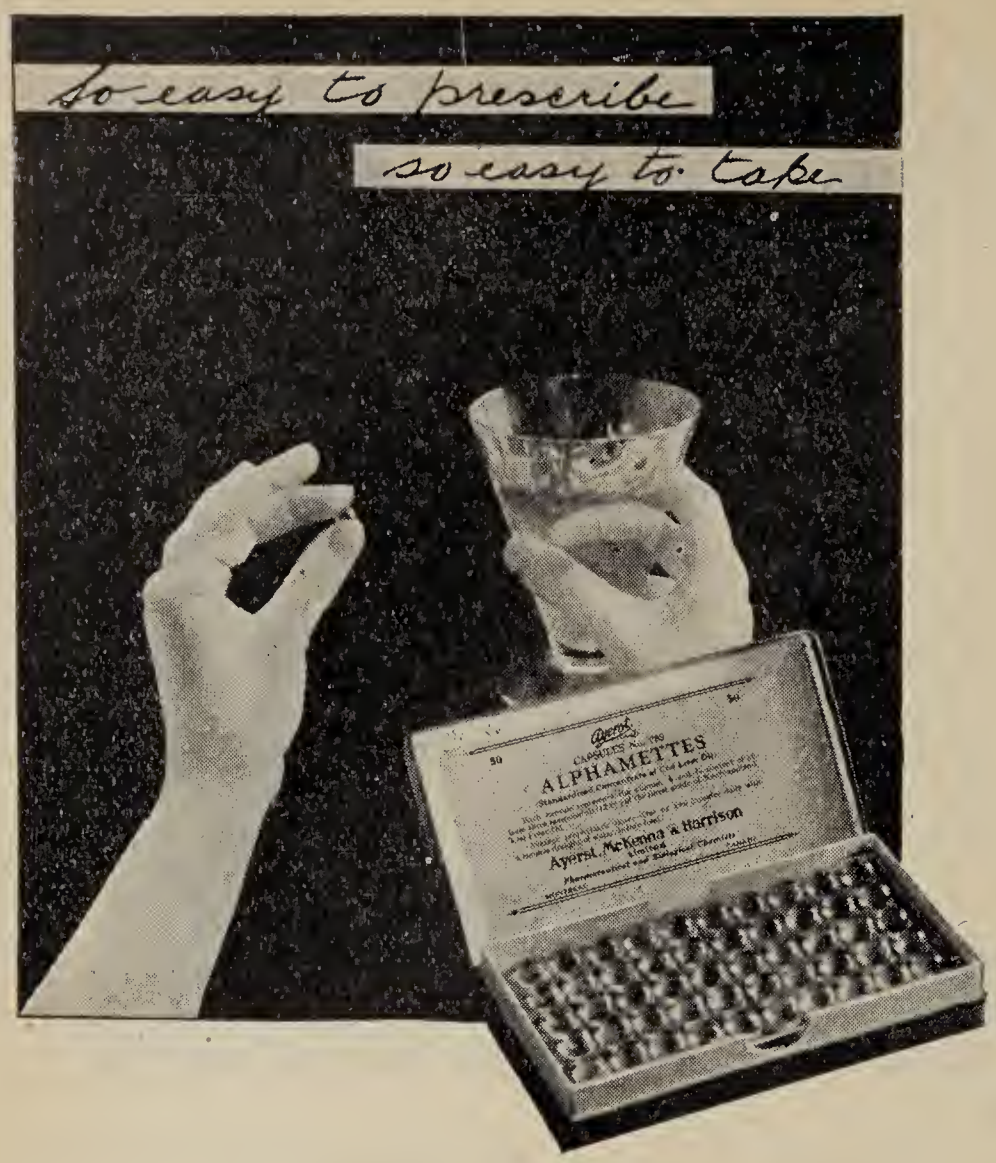

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